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Case Report

Observation of the development and spontaneous closure of a traumatic macular hole by optical coherence tomography

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Abstract

We report a case of a traumatic macular hole (TMH) formation which occurred several days after a blunt trauma in a child in order to demonstrate its spontaneous closure by daily optical coherence tomography (OCT) findings. A nine-year-old boy applied to the outpatient clinic with skin injury in the zygoma region which was caused by blunt trauma. Foveal contour was normal and subretinal and intraretinal fluid was detected on OCT. On the 2nd day, the subretinal fluid increased. On the 3rd day, there was a slight separation between the outer plexiform layer and the outer nuclear layer, suggesting a lamellar hole. A full-thickness macular hole with a base width of 360 microns was observed on the 7th day. On the 3rd week of follow–up, the OCT revealed closure of macular hole. A slight foveal depression and highly reflective accumulation and shadowing of retinal pigment epithelium were detected. A spontaneous closure may be observed in TMHs, especially in patients within the pediatric age group. Observation may be a better therapeutic option for TMHs before deciding on a macular surgery in childhood.

Keywords

Traumatic Macular Hole; Optical Coherence Tomography; Spontaneous Closure

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Introduction

Trauma is the second common cause of macular hole development after idiopathic macular holes. Ocular traumas are seen at any age, but more frequent at young ages and in males due to the activities such as sports and recreation [1]. Traumatic macular hole (TMH) is a well-known ocular entity, however, its mechanism is still unclear. There is no consensus on treatment in the literature. The aim of this study is to present the macular hole formation after several days of a blunt trauma in a child and to demonstrate its spontaneous closure by daily optical coherence tomography (OCT) findings.

Case Report

A nine-year-old boy referred to our outpatient clinic with a skin injury in the right zygoma region which was caused by a blunt trauma. The external examination revealed a 2 cm "Z" shaped multiple skin and subcutaneous incisions while lateral cantus was intact. There was no ocular motility disturbance. Orbital computerized tomography was normal. His best corrected visual acuity was 6/10, and intraocular pressure was 19 mmHg in the right eye. There were no abnormalities in the anterior segment examination other than scarce cells. Preretinal hemorrhage in an inferior macular region, subretinal hemorrhage near the optic disc, and scattered superficial retinal hemorrhages were detected upon fundus examination. The macular area was pale, suggestive of commotio retina. Optic disc was evaluated as normal. Fundus photography could not be taken since the patient did not cooperate. Ocular examination of the left eye was totally normal. On OCT of the right eye, foveal contour was normal and subretinal and intraretinal fluid was detected (Figure 1a). The wound was sutured on the same day. Topical treatment with nepafenac 0.1% 4x1, dexamethasone 0.1% 4x1, cyclopentolate hydrochloride 1% 3x1 were started postoperatively. Repeated OCT examination on the 2nd day of the accident revealed increased subretinal fluid (Figure 1b). On the 3rd day, there was a slight separation between the outer plexiform layer and the outer nuclear layer, suggesting a lamellar hole on OCT. Persistent subretinal fluid was detected (Figure 1c). Fundus examination revealed a crescent-shaped choroidal rupture near the temporal disc area, and a full-thickness macular hole with a base width of 360 microns was observed on OCT on the 7th day (Figure 1d). On the 3rd week of the follow-up, visual acuity was 10/10. Macular wrinkling, retinal pigment epithelium (RPE) hypertrophy and irregularity, improvement of retinal hemorrhages were detected on the fundus examination (Figure 2a). OCT revealed closure of macular hole. A slight foveal depression and a highly reflective accumulation and shadowing of RPE (consistent with RPE hypertrophy) were detected. RPE irregularity beneath the fovea and retinal thinning of the perifoveal area were observed (Figure 2b). Written informed consent was obtained from patients' parents who participated in this case.

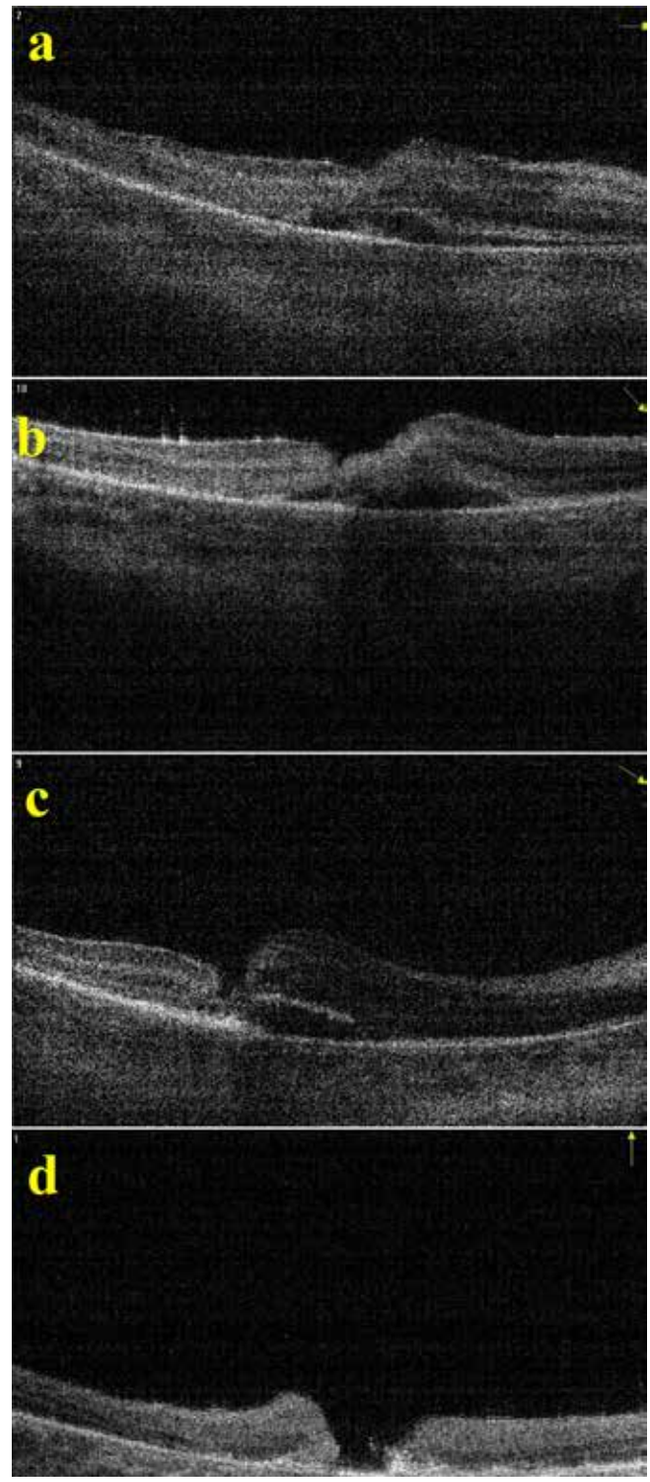

Figure 1. Macular OCT on the day of trauma: Foveal contour was normal and subretinal and intraretinal fluid was detected (a), OCT on the 2nd day after trauma: Increased subretinal fluid (b), OCT on the 3rd day after trauma: A slight separation between the outer plexiform layer and the outer nuclear layer, which was suggestive of lamellar hole (c), OCT on the 7th day after trauma: Full-thickness macular hole (d).

Discussion

The mechanism of the development of TMHs is still controversial. The researchers have put forward various theories to explain this entity. Yamashita et al. [2] proposed two different mechanisms depending on whether the posterior hyaloid is detached, or not. In the first mechanism, early (primary) separation of fovea leads to sudden visual loss. According to the second mechanism; foveal separation secondary to persistent vitreofoveal adhesion causes visual loss much later. Johnson et al. [3] suggested that sudden reduction in the anteroposterior diameter of the globe leads to compensatory expansion of the equatorial area, thus foveal retinal layers may split due to the flattening of the retina and tangential tractions. Yanagiya et al. [4] proposed that force of the stroke transmitted to macula on tight vitreoretinal attachments may result in foveal rupture. Delori et al. [5] suggested that rapid change in axial length due to

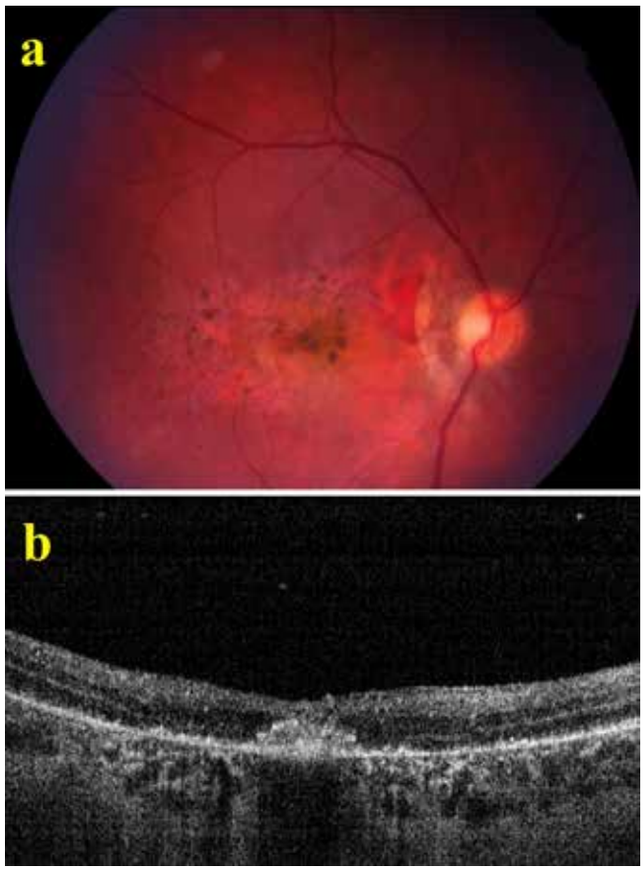

Figure 2. Fundus photograph on the 3rd week: Macular wrinkling, pigment epithelial hypertrophy, and irregularity, improvement of retinal hemorrhages(a), OCT on the 3rd week after trauma: Closure of hole, a slight foveal depression and a highly reflective accumulation and shadowing of RPE (consistent with RPE hypertrophy) (b).

trauma causes sudden air expansion that may result in macular hole formation. In children and young adults, the attachment between posterior hyaloid and the internal limiting membrane is stronger than in adults. Therefore, in children, the proposed mechanisms may generate a stronger effect on the macula inducing formation of TMHs more frequently.

Our case differs from the previously reported cases in that, although there was no macular hole immediately after trauma assessed by both clinical and OCT findings, we detected the TMH development during the follow-up period on the 7th day with the aid of daily OCT analysis. Commotio retina was suspected on fundus examination before the hole formation, accompanying intraretinal edema and subfoveal fluid seen on OCT at this earlier stage.

Although there are many studies reporting spontaneous recovery of TMHs, the mechanism is not fully understood. Some authors suggested that the development of epiretinal membranes leads to the contraction of the hole resulting in closure of the hole [6], others suggested RPE and glial cells adjacent to the macular hole proliferated and closed the hole [7], or total posterior hyaloid detachment facilitated hole closure by reducing the anteroposterior tractional forces [8]. On the other hand, there are some reports which deduced that the common feature of cases of spontaneous hole closure was the absence of development of posterior hyaloid detachment [2,9]. Other common features of spontaneously closed TMH cases are young age and smaller hole diameter [2,9,10].

In the case reported herein, there was no posterior hyaloid detachment on OCT neither before nor after trauma. Therefore, it may be thought that due to the attached posterior hyaloid, the effect of trauma was transmitted to the macula more intensely so that macular commotio occurred. The damaging effect of commotio on RPE may have impaired fluid transport towards the pigment epithelium from the retina leading to intraretinal and subretinal fluid accumulation. Through clinical examination and daily OCT analysis of this case, it may be speculated that the direct effect of commotio on retinal neural cells and/or the effect of commotio causing retinal fluid accumulation may have induced formation of a lamellar hole followed by the development of full-thickness hole.

In this case, spontaneous closure of the hole, as observed by OCT (Figure 2b), was thought to develop by the hypertrophic response of the RPE to commotio and that hypertrophy of the RPE played a part in the recovery of the macular hole. It was concluded that the young age of the patient may have had a role in the significant inflammatory response and RPE-cell hypertrophy that leads to spontaneous recovery of the hole.

Conclusion

Spontaneous recovery may be observed in TMHs, especially in patients who are within the pediatric age group. Therefore, TMHs in children must be followed with fundus examination, and OCT, for a more detailed and objective screening. RPE hypertrophy may play a role in the spontaneous closure of the hole in cases with commotio in children. Observation may be a better therapeutic option before deciding for macular surgery in childhood for TMHs.

Scientific Responsibility Statement

The authors declare that they are responsible for the article's scientific content including study design, data collection, analysis and interpretation, writing, some of the main line, or all of the preparation and scientific review of the contents and approval of the final version of the article.

Animal and human rights statement

All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. No animal or human studies were carried out by the authors for this article.

Conflict of interest

None of the authors received any type of financial support that could be considered potential conflict of interest regarding the manuscript or its submission.

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